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Summary

THE DUODENAL MICROENVIRONMENT IN FUNCTIONAL DYSPEPSIA SHOWS MINIMAL MOLECULAR AND FUNCTIONAL IMPACT ON THE INTESTINAL BARRIER AND MAST CELL BIOLOGY

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UEG Week Barcelona 2026 | Presentation

Mechanisms & Personalised Medicine | Small Intestine & Nutrition | Immunology | Endoscopy

October 20, 2026

Bulk RNA sequencing and proteomic profiling of duodenal biopsies from functional dyspepsia patients revealed only minor transcriptomic differences and no significant proteomic alterations in inflammatory markers compared to healthy controls, challenging the hypothesis of duodenal immune activation in FD.

KEY POINTS

- Only 42 genes were differentially expressed between 22 FD patients and 28 healthy controls, with 31 genes upregulated and 11 downregulated in FD; downregulated genes showed enrichment for TGF-β1 production (FDR: 0.008) and inflammatory response (FDR: 0.016), including reduced FURIN expression (log_2 fold change: -0.4, FDR: 0.02).
- Proteomic profiling of 96 inflammation-related protein biomarkers in duodenal biopsies from 29 FD patients and 29 healthy controls revealed no differences between the two cohorts.
- Principal component analysis showed no clear separation between FD and HC groups, highlighting the modest nature of transcriptional differences.
- Stimulation of HMC1.1 mast cells with biopsy supernatant from FD patients versus healthy controls did not result in significant differences in degranulation.
- Duodenal organoid-derived monolayers from both FD patients and healthy controls showed no significant change in permeability when stimulated with FD or HC biopsy supernatant for 48 hours, assessed by TEER, FITC-dextran (4kDa), and horseradish peroxidase passage.

– The findings argue against direct effector roles of locally secreted mediators in functional dyspepsia and do not support duodenal immune activation or barrier dysfunction as primary pathophysiological mechanisms.

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